

## Erysipeloid – Case Report

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**SUMMARY** Erysipeloid is an acute, bacterial infection of traumatized skin in an individual who was in direct contact with meat or other animal products contaminated with a gram-positive bacillus *Erysipelothrix rhusiopathiae*. We present a case of a 50-year-old housewife whose hobby was fishing, with a reddish, tender patch on the fifth finger and dorsum of the left hand, which developed a week after she had sustained an injury while boning the fish. The patient was treated with orally administered penicillin V 1,500,000 IU *t.i.d.* for 7 days, with complete resolution.

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## INTRODUCTION

Erysipeloid is an acute, bacterial infection of traumatized skin in an individual who was in direct contact with meat or other animal products contaminated with a gram-positive bacillus *Erysipelothrix (E.) rhusiopathiae* (1-3). In 1878, *E. rhusiopathiae* was first isolated by Koch (4). The term "erysipeloid" was first used by Rosenbach, in order to distinguish it from human erysipelas (5). In 1909, he designated *E. rhusiopathiae* as a human pathogen (5).

## CASE REPORT

A 50-year-old woman visited Outpatient Clinic at University Department of Dermatology and Venereology, Zagreb University Hospital Center for tender and painful purple-red papules on the dorsum of her left hand. The lesion developed a week after she had been injured while boning the fish. The injured site was on the fifth finger of the left hand. Initially she felt burning pain limited to the

site of minor injury. Shortly after, reddish papules appeared, sparing the terminal phalange of the fifth finger (Fig. 1). Gradually, the lesion spread peripherally to the dorsal side of the left hand. The adjacent joint turned painful and swollen. As the sharply delineated edge was advancing peripherally, the center was clearing and the patient experienced discrete sensation of itching. Generally, she was healthy, without constitutional symptoms like fever and malaise, and without pathologic laboratory findings. Culture of lesion biopsy was not done because the patient's medical history and clinical findings suggested the diagnosis. The patient was treated with orally administered penicillin V 1,500,000 IU *t.i.d.* for 7 days. As the lesion was resolving, brownish discoloration developed and disappeared shortly after therapy discontinuation (Fig. 2). Soon after penicillin administration, her lesion completely resolved and there was no recurrence.



**Figure 1.** A red, partly violaceous, slightly edematous papular eruption with a well raised edge, with central regression on the back of the hand before therapy.

## DISCUSSION

The causative agent, *E. rhusiopathiae* (formerly known as *E. insidiosa*) is a thin, rod-shaped, facultative anaerobic, non-acid-fast, nonsporulating, and nonmotile, gram-positive bacillus. The organism is resistant to environmental influences such as salting, smoking, pricking, exposure to direct sunlight for 12 days, and may stay alive for many months in carcasses (1,2). The organism may live long enough to cause infection weeks or months after initial soil contamination (1). It is widely distributed among many wild and domestic animals such as fish, pigs, sheep, poultry, cattle, etc. However, it is mostly found in pigs, causing a disease known as swine erysipelas (1,3,6-8). The etiologic agent of swine erysipelas was described by Loeffler in 1886 (6). *E. rhusiopathiae* can also be isolated from fresh and seawater fish and crustaceans, but in fish itself it causes no known disease (1,9). Erysipeloid can also be transmitted by contaminated food (1,3).

Erysipeloid is almost always an occupational disease. The disease is associated with a variety of occupations among which fishermen and fish retailers, butchers, farm workers, veterinarians, housewives, and in the past also button (bone) and leather workers as well as soap makers are at highest risk (1-3,7,9). Connor *et al.* have described coexistence of erysipeld and orf in sheep farmer (10). Our patient got infected by handling the fish, while boning it. She was a housewife whose hobby was fishing.

The disease can affect any race, age and sex group. According to professional exposure, the in-



**Figure 2.** Erysipeloid patient 7 days after penicillin therapy.

cidence is higher in males (2,3,7,8). The incubation period is 2 to 7 days or 2 weeks at the most. Erysipeloid can be divided into localized cutaneous form, diffuse cutaneous form, and systemic infection complicated with endocarditis, or joint, bone, brain and pleural involvement (3,7,9,11). According to Fitzpatrick *et al.* it can be divided into four different forms: (a) localized cutaneous (also known as erysipeld of Rosenbach); (b) diffuse cutaneous; (c) subacute bacterial endocarditis; and (d) systemic infection without endocarditis, usually in immunocompromised individuals (2).

The localized form is most frequently found in humans. The lesions are usually confined to the site of inoculation, such as a finger or hand, but any site can be involved. The primer lesion is well-defined reddish to violet papule, which can sometimes show confluence into patches. They gradually become swollen, warm and tender, accompanied by burning and throbbing sensation. Adjacent joints are often involved, and are swollen and painful. As the patches spread peripherally, the central zone clears. Sometimes hemorrhagic vesicles and bullae without suppuration may appear. In some patients lymphangiitis and lymphadenopathy may occur, while systemic symptoms such as low-grade fever and malaise are uncommon (1,2). As the lesions resolve, brownish discoloration may develop (2,4,7,11). Rarely multiple, serpiginous, violaceous, widespread cutaneous lesions appear distantly from the site of injury. The course of disseminated cutaneous form is protracted with the possibility of recurrences (1-3,7,8,11). Subacute endocarditis is the most common complication of the rare systemic infection. There is a great predisposition for involvement of the aortic valve, which was previously normal in nearly 60% of cases (1). In case of systemic in-

fection, the patient may have a typical local cutaneous lesion or that with central necrosis, or may have widespread lesions ranging from perifollicular papules to purpuric exanthema and necrosis, but usually without cutaneous manifestation. Other reported manifestations of systemic infection are septic arthritis, diffuse glomerulonephritis, meningitis, conjunctivitis, bronchitis, pleural and bone involvement (1-3,7,11). Our patient had a cutaneous localized erysiploid. Her site of inoculation was the fifth finger of the left hand, presented with reddish to violet papule that showed confluence into the plaque spreading to the dorsal side of the left hand (Fig. 1). She experienced burning pain accompanied by itching sensation.

The diagnosis of cutaneous disease is made on the basis of clinical findings and medical history. Actually it is a cellulitis because the causative agent is situated in deeper skin layers. Culture of the lesion biopsy specimen has to be performed from the edge of the lesion and it should contain full thickness of the dermis (1,2,12). The causative agents are rarely revealed from the surface of the lesion or from aspirated material (1-3,11). Identification is based on Gram stain. Sometimes, like in atypical infections, Gram stain can be misidentified as *Lactobacillus* spp. or *Enterococcus* spp., so the identification schemes should include testing for hydrogen sulfide ( $H_2S$ ) production (1,2,12). Commercial blood culture media are available for detection in systemic infection. Blood culture is negative in both cutaneous forms. Echocardiography, computed tomography (CT) or brain magnetic resonance imaging (MRI), chest radiography or CT, a scan or MRI of bones may be performed to aid in the diagnosis of some systemic manifestations (1-3,8).

Differential diagnosis includes erysipelas and other forms of bacterial cellulitis, spider bites, fixed drug eruptions, and erythema migrans. The course of erysipelas is rapid, most patients are very ill, with elevated white blood cell (WBC) and erythrocyte sedimentation rate (ESR); central area remains the most affected part of the lesion and there is no spontaneous resolution (2,3,7,11). The disease is self-limited and regresses spontaneously within 2 to 4 weeks (1-3). However, if not treated, the disease may persist for months (1).

The mainstay of therapy are antibiotics. The drug of choice is penicillin; in penicillin-allergic patients imipenem, ciprofloxacin, and erythromycin can be administered. Resistance to erythromycin, tetracycline, and chloramphenicol has been described (1-3,7,11,13). Our patient was treated

with orally administered penicillin V 1,500,000 IU t.i.d. for 7 days. After antibiotic discontinuation, the lesions completely resolved. Various disinfectants show activity against *E. rhusiopathiae* (14). To prevent the spread of the disease, it is advised to use gloves while handling raw fish, meat and other animal products (7). The cutaneous form of infection has a favorable prognosis, however, in systemic forms the course and prognosis depend on organ involvement as well as on early and appropriate treatment (2,3,8). After erysiploid infection, no permanent immunity develops (2,3,15,16). A vaccine for veterinarians and animal handlers is available, but the efficacy is questionable (3).

## CONCLUSION

Erysipeloid is a rare bacterial disease, and it is almost always seen in persons handling contaminated animal products. Although it is mostly self-limited, antibiotic therapy is suggested to avoid the potential second attacks and disease persistence. It is considered as an occupational disease, so appropriate preventive measures should be taken.

## References

1. Reboli AC, Farrar WE. *Erysipelothrix rhusiopathiae*: an occupational pathogen. Clin Microbiol Rev 1989;2:354-9.
2. Swartz MN, Weinberg AN. Miscellaneous bacterial infection with cutaneous manifestations. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, editors. Fitzpatrick's dermatology in general medicine. 6<sup>th</sup> ed. New York (NY): McGraw-Hill; 2003. p. 1921-2.
3. Braun-Falco O, Plewig G, Wolff HH, Burgdorfer WH. Erysipeloid. In: Braun-Falco O, Plewig G, Wolff HH, Burgdorfer WH, editors. Dermatology. 2<sup>nd</sup> completely revised ed. Berlin: Springer Verlag; 2000. p. 193-4.
4. Koch R. Investigations into the etiology of traumatic infective disease. London: New Sydenham Society, 1880.
5. Rosenbach FJ. Experimentelle morphologische und klinische Studie über die Krankheits-erregenden Mikroorganismen des Schweine-rotlaufs, des Erysipeloids und der Mausepsie. Z Hyg Infektionskr 1909;63:343-69.
6. Loeffler FA. Experimentelle Untersuchungen über Schweinerotlauf. Abs Kais Gesundheitssamte 1886;1:46-55.

7. Blume JE, Levine EG, Heymann WR. Bacterial diseases. In: Bologna JL, Jorizzo JL, Rapini RP, Horn TD, Mascaro JM, Mancini AJ, Salasche SJ, Saurat J-H, Stengl G, editors. Dermatology. Edinburgh: Mosby; 2003. p. 1130.
8. Ghorayeb ZN, Matta-Muallem M. Erysipeloid. *eMedicine J* 2005;1-9. Available at: <http://www.emedicine.com/derm/topic602.htm>. Accessed: December 14, 2005.
9. Fidalgo SG, Wang Q, Riley TV. Comparison of methods for detection of *Erysipelothrix* spp. and their distribution in some Australian seafoods. *Appl Environ Microbiol* 2000;66:2066-70.
10. Connor MP, Green AD. Erysipeloid infection in a sheep farmer with coexisting orf. *J Infect* 1995;30:161-3.
11. Hay RJ, Adriaans BM. Bacterial infections. In: Burns T, Breathnach S, Cox N, Griffiths C, editors. *Rook's textbook of dermatology*. Turin: Blackwell Science Ltd; 2004. p. 27-43.
12. Dunbar SA, Clarridge JE 3<sup>rd</sup>. Potential errors in recognition of *Erysipelothrix rhusiopathiae*. *J Clin Microbiol* 2000;38:1302-4.
13. Venditti M, Gelfusa V, Tarasi A, Brandimarte C, Serra P. Antimicrobial susceptibilities of *Erysipelothrix rhusiopathiae*. *Antimicrob Agents Chemother* 1990;34:2038-40.
14. Fidalgo SG, Longbottom CJ, Riley TV. Susceptibility of *Erysipelothrix rhusiopathiae* to antimicrobial agents and home disinfectants. *Pathology* 2002;34:462-5.
15. Shimoji Y. Pathogenicity of *Erysipelothrix rhusiopathiae*: virulence factors and protective immunity. *Microbes Infect* 2000;2:965-72.
16. Shimoji Y, Ogawa Y, Osaki M, Kabeya H, Maruyama S, Mikami T, *et al.* Adhesive surface proteins of *Erysipelothrix rhusiopathiae* bind to polystyrene, fibronectin, and type I and IV collagens. *J Bacteriol* 2003;185:2739-48.



With Nivea cream on the fresh air and sun, year 1934.  
(from the collection of Mr. Zlatko Puntijar)